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Correlation between occlusal contact and root resorption in teeth with periodontal disease

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Background and Objective: The present study was designed to investigate the influence of occlusal forces on radicular resorption in teeth with periodontal disease. The occlusal forces are a cause in the aggravation of the periodontal disease and therefore influences in the increase the extension and the depth of the radicular resorption.

Material and Methods: We quantified radicular resorption, its extension across the radicular surface and its depth, in 88 teeth with periodontal disease with and without occlusal contact, pertaining to patients between 43 and 91 years of age. A histological method was used to obtain 6-μm-thick sections. The sections were observed under an Olympus BX40 optical microscope and processed by an image analysis program. Measurements of length and area were used to calculate the percentages of surface and volume of cement reabsorbed.

Results: In both groups presenting periodontal pathology (groups 2 and 3) the percentages of the surface and volume of reabsorbed cement were greater in those teeth with antagonist contact. The greatest percentages of radicular resorption were observed in teeth of group 3 showing antagonism.

Conclusion: The severity of periodontal disease increases the extension and the depth of the radicular resorption, and the presence of antagonist forces aggravates the resorption.

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Radicular resorption has been defined as a physiological process that drives active remodelling of the cementum and dentine (1). In contrast, we refer to radicular resorption in permanent teeth to describe a pathological process with clinical and radiological manifestations that produce destruction of the tooth root (2). In healthy permanent teeth, resorption is superficial, painless and discontinuous, and repairs rapidly. It is most frequently located in the apical third (3–5). Radicular resorption and reduction in reparative capacity are related to severity of periodontal disease (5), but also affect healthy teeth subjected to excessive forces, such as those that occur during orthodontic treatment. Orthodontic forces applied produce an inflammatory process that favours tooth movement within the alveolus (4,6); the magnitude, duration and type of applied force can cause resorption of the hard tissue and root shortening (1,6–9). The periodontal ligament is an absolute requirement for remodelling the alveolar bone when physical forces are applied to teeth (10). Physiological loading or orthodontically induced tooth movements involve remodelling of the periodontal tissue, and an equilibrium exists between the resorption and the formation of cementum.

In teeth with reduced support, implying alteration of the ligament, as occurs in periodontal disease, the stress during mastication becomes excessive. When this combines with an increased movement of the tooth in the alveolus, it brings about an alteration in the balance of the relation between resorption and formation of cementum. We maintain that this aggravates the destruction of the radicular cement.

The aim of the present study was to investigate the influence of occlusal forces on the radicular resorption in teeth with periodontal disease. The study therefore aimed to quantify the radicular resorption in teeth with and without occlusal contact.

Material and methods

For this study, eighty-eight teeth (43 premolars and 45 molars) with periodontal disease were used. A clinical history was obtained prior to extraction, and medically compromised patients were excluded from the study. Patients who had received periodontal treatment or those with signs of pulpar disease were also excluded from the study. The patients received written and oral information on the study. Informed consent was obtained from each patient after explaining the procedures.

Before tooth extraction, a periapical radiograph was taken using intraoral radiographic equipment (65 kV). Probing depth and attachment loss were measured and compared with radiographs. All teeth showed an insertion loss (line distance from the cemento-enamel junction to the bottom of the pocket) of a least 4 mm at any place, a degree of mobility Grade II and bleeding on probing. Teeth were divided into the following two groups according to previously established radiographic criteria (5,17): group 2, teeth with bone loss between one- and two-thirds of the normal alveolar height; and group 3, teeth with bone loss greater than two-thirds of the normal alveolar height. In group 2 (n = 30), 14 teeth presented with antagonist contact, while in group 3 (n = 58), 30 teeth presented with occlusal contact.

Previous studies have shown (5) that histological changes, specifically root resorption, in teeth with periodontal disease group 1 were repaired. Therefore, extraction of a group 1 tooth would not be justified in clinical practice. This is why we decided to analyse only teeth which could be included in groups 2 and 3 in order to investigate the influence of antagonistic forces on the aggravation of radical resorption in teeth with periodontal disease.

After the teeth had been extracted, they were washed in phosphate-buffered saline solution and fixed in 4% buffered formaldehyde for 48 h, after which they were decalcified in Osteodec (Bio.optic) for 20–30 d. Each tooth was then sectioned into two halves in the mesiodistal direction, dehydrated in alcohol, and then cleansed in toluene and embedded in paraffin. Serial sections of 6 μm thickness were obtained and collected on slides; five sections per slide, and 20 slides for half a tooth (40 slides per tooth). The sections were stained with haematoxylin and eosin and Mason's trichrome. An average of 200 sections (1200 μm per tooth) were analysed with an Olympus Bx40 microscope. One in every three sections was photographed (400 μm per tooth) with an Olympus DP 10 camera connected to the microscope. The image was then analysed with a software program (MICRO IMAGE 3.0., Hamburgo, Alemania).

Morphometric study

Resorption was measured in sections in which the root is observed in all his length. In biradicular premolars, buccal and palatal roots were measured; in superior molars, mesiobuccal roots, distobuccal and palatine roots were measured; and in lower molars, mesial and distal roots were measured. In the sections observed with the Olympus Bx40 microscope, the superior limit of the cementum was marked on the coverslip to measure the total length of the root of every tooth and the total area of the root. Subsequently, four independent measurements were taken using the computer program; the length and surface area in micrometres of the total of the radicular cementum and of cementum resorption. The data were grouped and then entered into a Microsoft Excel table. Figures 1 and 2 show examples of mathematical calculations performed to obtain the percentages of surface and volume of cementum resorbed.

Statistical analysis

The measurements of the surface and volume reabsorbed are presented with the mean values and respective standard deviations. T-Student was used to compare the measurements between the two different groups, and ANOVA was used for comparisons of more than two groups. Bonferroni methodology was also used for subsequent comparisons. In all cases, a result was considered significant where $p < 0.05$.

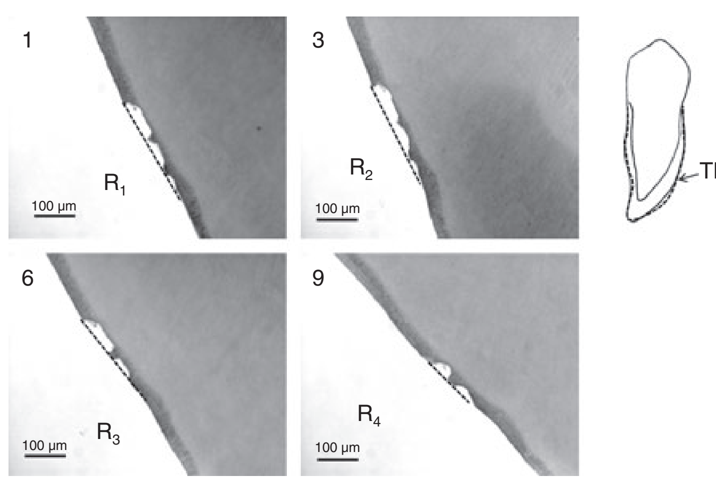

Fig. 1. Resorption in the middle third of the root of a premolar in group 2 shown in several serial sections (1,3,6,9). (Magnification ×10). RL (average length) = R1 + R2 + R3 + R4 / 4 ; TL (total length) ;snr (sections number with resorption); in the example, snr = 9 ; RS (reabsorbed surface) = LR X snr X 6 μm; sn (sections number) = 200 ± 20; TS (total root surface) = TL (total length) X sn X 6 μm; % RS = RS / TS x 100.

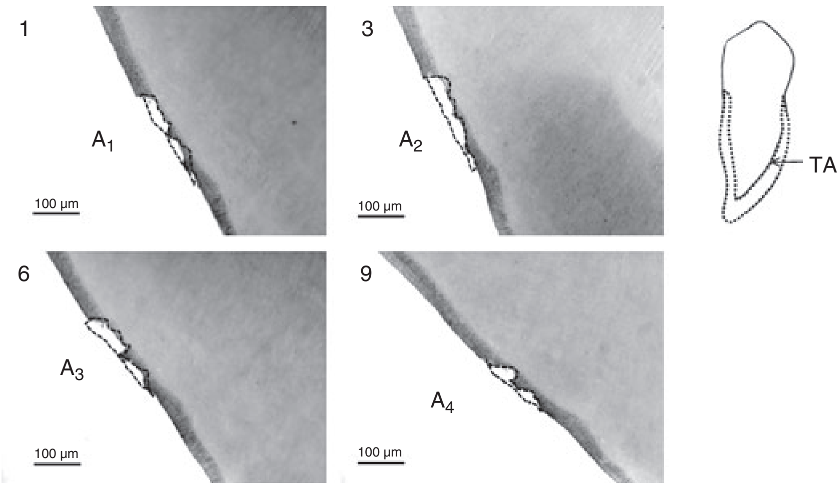

Fig. 2. Resorption in the middle third of the root of a premolar in group 2 shown in several serial sections (1,3,6,9). (Magnification ×10). RA (reabsorbed area) = A1 + A2 + A3 + A4 /4 ; TA (total area); snr (sections number with resorption); in the example, snr = 9 ; RV (reabsorbed volume) = RA X snr X 6 µm; sn (sections number) = 200 ± 20; TV (total cementum volume) = TA (total area) X sn X 6 µm; %RV = RV / TV x 100.

Results

Radicular resorption was found in all the teeth studied. Teeth with periodontal disease in group 3 showed significantly higher values both for surface ($p = 0.036$) and volume resorption ($p = 0.009$) than teeth with periodontal disease in group 2 (Figs 3 and 4 and Table 1).

The percentage of reabsorbed surface and volume was significantly greater in teeth which showed an antagonist compared with teeth without any antagonist ($p = 0.009$ and $p = 0.003$, respectively; Figs 5 and 6 and Table 2).

We find the highest percentage of reabsorbed surface and volume in teeth with an antagonist and group 3 periodontal disease.

In teeth presenting with an antagonist, those with group 3 periodontal disease showed a higher value than those in group 2; this difference was only significant in the percentages of reabsorbed volume ($p = 0.032$).

In teeth without an antagonist, those showing group 3 periodontal disease also presented higher percentages of surface and volume compared with group 2, but these differences were not significant.

When the four groups were compared together, there was a significant difference in the percentages of reabsorbed surface in teeth with an antagonist and showing group 3 periodontal disease, in contrast to those teeth without anantagonist both in group 3 ($p = 0.031$) and in group 2 ($p = 0.038$). The percentages of reabsorbed volume showed a significant difference between teeth with an antagonist and group 3 periodontal disease compared with the other teeth; i.e. when compared with those showing an antagonist and group 2 disease (($p = 0.032$), when compared with teeth without an antagonist in periodontal disease group 2 ($p = 0.011$), and finally in comparison with teeth without an antagonist and with group 3 periodontal disease: ($p = 0.002$; Figs 7–9 and Tables 3 and 4).

Discussion

The majority of the literature about radicular resorption in the permanent teeth relates to orthodontic cases. Studies regarding the presence of resorption in teeth with adult periodontitis are few (5,11–17).

Molar and premolar teeth were selected for use in the present study. In human dentition, the molars and premolars do the most of the masticatory work, but are helped by the canines. However, as the canines do not possess such a large an occlusal surface as the molars they participate less in mastication (18). Both in these theoretical models and in experiments *in vivo*, the impact of the forces of mastication is larger in the posterior sector, intermediate in the canines and smaller in the incisors (19–22).

In our study, we observed that radicular resorption was greater both in extension (surface) and in depth (volume) when there was an antagonist tooth, in both groups of periodontal disease. Furthermore, the greater

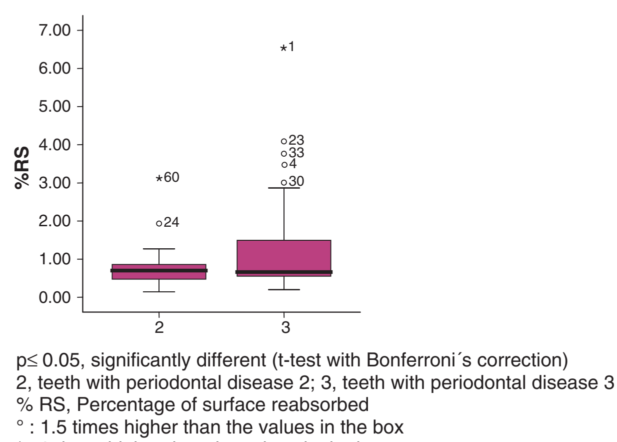

Fig. 3. Percentage of surface reabsorbed (%RS) according to the severity of periodontal disease. p ≤ 0.05, significantly different (t-test with Bonferroniś correction); 2, teeth with periodontal disease 2; 3, teeth with periodontal disease 3; % RS, Percentage of surface reabsorbed; ° : 1.5 times higher than the values in the box; * : 3 times higher than the values in the box.

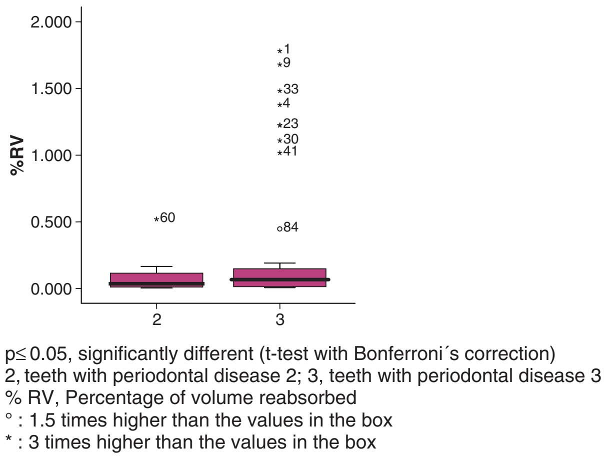

Fig. 4. Percentage of volume reabsorbed (%RV) according to the severity of periodontal disease. p ≤ 0.05, significantly different (t-test with Bonferronís correction); 2, teeth with periodontal disease 2; 3, teeth with periodontal disease 3; % RV, Percentage of volume reabsorbed; ° : 1.5 times higher than the values in the box; * : 3 times higher than the values in the box.

Table 1. Percentage of volume and surface area reabsorbed by the severity of periodontal disease

	n	Mean	SD	*p*-value
%RS				
Grade 2	30	0.796	0.5818	$p = 0.036$*
Grade 3	58	1.193	1.16548	
%RV				
Grade 2	30	0.0769	0.10091	$p = 0.009$*
Grade 3	58	0.2481	0.46435	

Grade 2, teeth with periodontal disease grade 2; grade 3, teeth with periodontal disease grade 3; %RS, percentage of surface reabsorbed; and %RV, percentage of volume reabsorbed.
*$p \leq 0.05$, significantly different (T-Student).

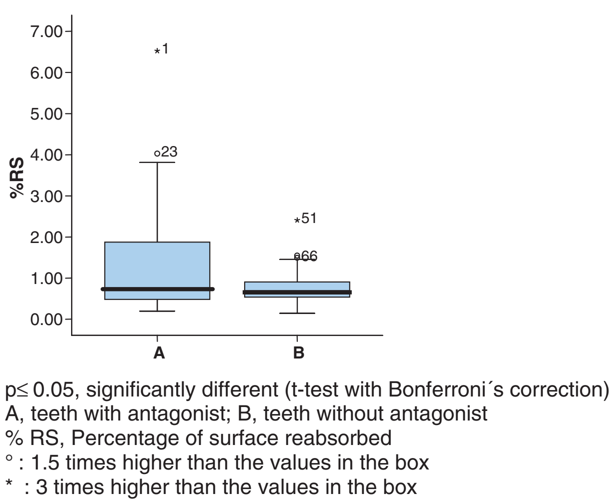

Fig. 5. Percentage of area reabsorbed (%RS) by the presence or absence of antagonist. p ≤ 0.05, significantly different (t-test with Bonferronís correction); A, teeth with antagonist; B, teeth without antagonist; % RS, Percentage of surface reabsorbed; ° : 1.5 times higher than the values in the box; * : 3 times higher than the values in the box.

depth of resorption was significant when both factors (the severity of the disease and the presence of antagonists) combined. Although there are no studies that analyse the influence of occlusal contact on the resorption of the radicular cement in teeth with periodontal disease, there are numerous studies regarding the effects of masticatory forces on teeth.

In healthy teeth, the masticatory forces from antagonist teeth stimulate the process of remodelling of not only the cementum but also the alveolar bone (23), which is more susceptible to resorption than radicular cementum. It is known that the masticatory function stimulates bone resorption (24), and the absence of function stimulates bone formation (1,7,25,26,27) and increases the risk of dental ankylosis in implanted teeth (25,28). Although functional stimulation is necessary for the maintainence of the tissues, mastication can end up by producing potentially traumatic tooth movements for the periodontium (29).

In orthodontic treatments, the force applied can cause resorption of the alveolar bone in the areas of pressure and reduce the amount of radicular cement and the dentine (6,26,30,31). Orthodontic movement of teeth activates inflammatory mediators involved in the process of tissue resorption (32–43). In healthy teeth, resorption is followed by repair, and periodontal health is maintained. By contrast, in teeth with periodontal disease the occlusal trauma increases the destruction of periodontal tissue (10,44–49).

In our study, we found that all the teeth, especially those in contact with an antagonist, were submitted to excessive movements due to loss of insertion. This means that even with a normal chewing pattern, there is greater displacement of the tooth in the socket, with greater impact on the periodontal ligament. Our results, in agreement with previous studies (5,17,50), showed that without considering the presence of antagonists, there was greater radicular resorption in teeth with group 3 periodontal disease than in those of group 2. When we compared teeth with and without antagonists, we found that resorption

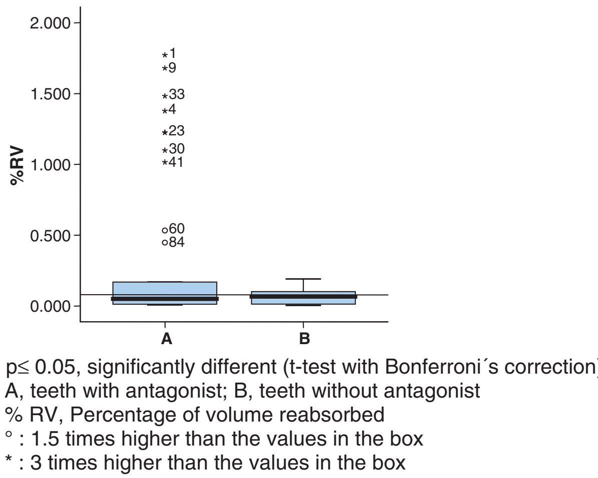

Fig. 6. Percentage of volume reabsorbed (%RV) by the presence or absence of antagonist. p ≤ 0.05, significantly different (t-test with Bonferroni´s correction); A, teeth with antagonist; B, teeth without antagonist; % RV, Percentage of volume reabsorbed; ° : 1.5 times higher than the values in the box; * : 3 times higher than the values in the box.

Table 2. Percentages of volume and surface area reabsorbed by the presence or absence of antagonist

	n	Mean	SD	*p*-value
%RS				
A	44	1.340	1.32332	$p = 0.009$*
B	44	0.7748	0.43155	
%RV				
A	44	0.31520	0.52072	$p = 0.003$*
B	44	0.06434	0.05306	

A, teeth with antagonist; B, teeth without antagonist; %RS, percentage of surface reabsorbed; and %RV, percentage of volume reabsorbed.
*$p \le 0.05$, significantly different (T-Student).

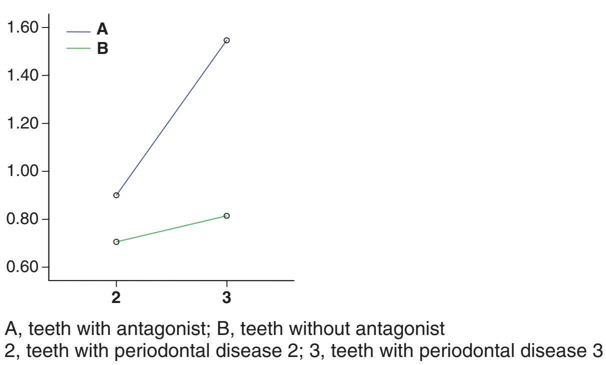

Fig. 7. Percentage of surface reabsorbed according to periodontal disease and the presence or absence of antagonist. A, teeth with antagonist; B, teeth without antagonist; 2, teeth with periodontal disease 2; 3, teeth with periodontal disease 3.

increased when there was occlusal contact. In teeth without an antagonist, there was no significant difference between the two groups of periodontal disease, whereas in teeth with an antagonist the volume of reabsorbed cementum was greater in teeth belonging to group 3. The greatest resorption, with a significantly different value, was in teeth of group 3 with an antagonist. Our results show that in teeth with periodontal disease, occlusal contact is an aggravating factor in radicular resorption.

This finding is consistent with other studies showing that excessive occlusal forces may be a risk factor of periodontal destruction (49), so subjects with reduced periodontum have greater holding forces between opposing incisors (51) and periodontal destruction is associated with increased biting pressure (52). Antagonistic forces that would not be excessive for a healthy periodontum are harmful in a diseased periodontum.

Clinical relevance

Teeth with and without antagonists were extracted from patients with periodontal disease and analysed to assess the influence of occlusal contact on the presence and severity of root resorption. Contact with an antagonist enhances root resorption in teeth with periodontal disease. Our results suggest that opposing forces in teeth with attachment loss are an aggravation of periodontal disease. This work helps to confirm that during treatment of periodontal disease it is necessary to avoid or minimize the occlusal forces of antagonists.

References

1. Brudvik P, Rygh P. The initial phase of orthodontic root resorption incident to local compression of the periodontal ligament. *Eur J Orthod* 1993;**15:**249–263.
2. Darendeliler MA, Kharbanda OP, Chan EK *et al.* Root resorption and its association with alterations in physical properties, mineral contents and resorption craters in human premolars following application of light and heavy controlled orthodontic forces. *Orthod Craniofac Res* 2004;**7:**79–97.
3. López NJ, Gioux C, Canales ML. Histological differences between teeth with adult periodontitis and prepubertal periodontitis. *J Periodontol* 1991;**61:**87–94.
4. Bosshardt DD, Selvig KA. Dental cementum: the dynamic tissue covering of the root. *Periodontol 2000* 1997;**13:**41–75.
5. Crespo Abelleira A, Rodríguez Cobos A, Fuentes Boquete I, Castaño Oreja MT,

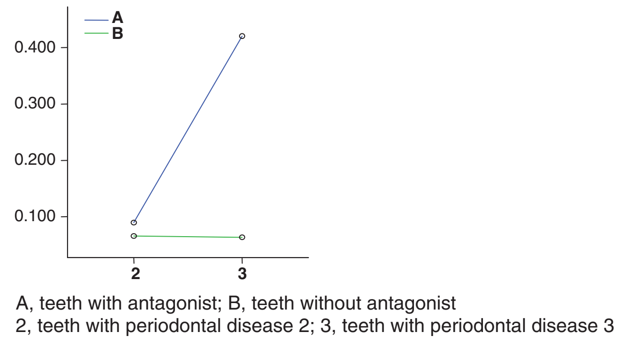

Fig. 8. Percentage of volume reabsorbed according to periodontal disease and the presence or absence of antagonist. A, teeth with antagonist; B, teeth without antagonist; 2, teeth with periodontal disease 2; 3, teeth with periodontal disease 3.

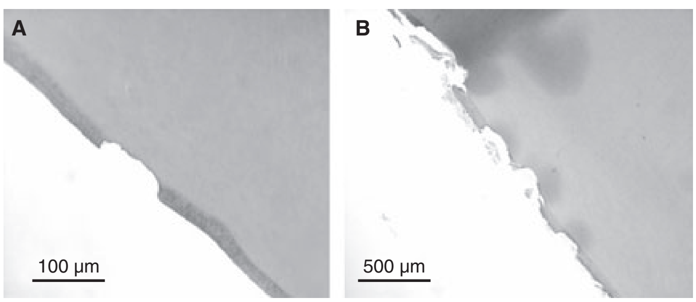

Fig. 9. (A) Resorption area in the middle third of a group 2 premolar. (B) Middle third of a group 3 premolar with oclussal contact.

Table 3. Percentages of reabsorbed area according to the severity of periodontal disease and the presence or absence of antagonist

(I)	(J)	Difference between means (I–J)	SEM	*p*-value
A2	B2	0.1940	0.35535	NS
	A3	−0.6466	0.31428	NS
	B3	0.0856	0.31784	NS
B2	A3	0.8406*	0.30059	$p = 0.038$*
	B3	−0.1084	0.30430	NS
A3	B3	0.7322*	0.25515	$p = 0.031$*

2, teeth with periodontal disease grade 2; 3, teeth with periodontal disease grade 3; A, teeth with antagonist; B, teeth without antagonist; and NS, not significant.
*$p \leq 0.05$, significantly different (*t*-test with Bonferroni's correction).

Table 4. Percentages of reabsorbed volume according to the severity of periodontal disease and the presence or absence of antagonist

(I)	(J)	Difference between means (I –J)	SEM	*p*-value
A2	B2	0.02385	0.130834	NS
	A3	−0.33077*	0.115714	$p = 0.032$*
	B3	0.02619	0.117022	NS
B2	A3B3	−0.35461*	0.110673	$p = 0.011$*
		0.00234	0.112040	NS
A3	B3	0.35696*	0.093942	$p = 0.002$*

2, teeth with periodontal disease 2; 3, teeth with periodontal disease 3; A, teeth with antagonist; B, teeth without antagonist; and NS, not significant.
*$p \leq 0.05$, significantly different (*t*-test with Bonferroni's correction).

Jorge Barreiro FJ, Rodríguez Pato R. Morphological study of root surfaces in teeth with adult periodontitis. *J Periodontol* 1999;**70:**1283–1291.

6. Brezniak N, Wasserstein A. Orthodontically induced inflammatory root resorption. Part I: the basic science aspects. *Angle Orthod* 2002;**72:**175–179.
7. Owman-Moll P, Kurol J, Lundgren D. Effects of a doubled orthodontic force magnitude on tooth movement and root resorptions. An inter-individual study in adolescents. *Eur J Orthod* 1996;**18a:** 141–150.
8. Owman-Moll A. The effects of a four-fold increased orthodontic force magnitude on toth movement and root resorptions. An intra-individua study in adolescents. *Eur J Orthod* 1996;**18b:**287–294.
9. Kurol J, Owman-Moll P, Lundgren D. Time-related root resorption after application of a controlled continuous orthodontic force. *Am J Orthod Dentofacial Orthop* 1996;**110:**303–310.
10. McCulloch CA, Lekic P, McKee MD. Role of physical forces in regulating the form and function of the periodontal ligament. *Periodontol 2000* 2000;**24:**56–72.
11. Henry JL, Weinmamm JP. The pattern of resorption and repair of human cementum. *J Am Dent Assoc* 1951;**42:**270–290.
12. Harvey J, Zander H. Root surface resorption of periodontally diseased teeth. *Oral Surg Oral Med Oral Pathol* 1959;**12:**1439–1443.
13. Sottosanti JS. A possible relationship between occlusion, root resorption, and the progression of periodontal disease. *J West Soc Periodontol Periodontal Abstr* 1977;**25:**69–74.
14. López NJ, Gigoux C, Canales ML. Morphologic and histochemical characteristics of the dental cuticle in teeth affected by prepubertal periodontitis. *J Periodontol* 1990;**61:**95–102.
15. Douglass KD, Cobb CM, Berkstein S, Killoy WJ. Microscopic characterization of root surfaceassociated microbial plaque in localized juvenile periodontitis. *J Periodontol* 1990;**61:**475–484.
16. Ökte E, Unsal B, Bal B, Erdemli E, Akbay A. Histological assesment of root cementum at periodontally healthy and diseased human teeth. *J Oral Sci* 1999;**41:**177–180.
17. Rodríguez Pato R. Root resorption in chronic periodontitis: a morphometrical study. *J Periodontol* 2004;**75:**1027–1032.
18. Ash MM, Nelson SJ. *Wheeler Anatomía, Fisiología y oclusión dental*. Madrid: 8ª ed. Elsevier España, 2004.
19. Dean JS, Throckmorton GS, Ellis E 3rd, Sinn DP. A preliminary study of maximum voluntary bite force and jaw muscle efficiency in pre-orthognathic surgery patients. *J Oral Maxillofac Surg* 1992; **50:**1284–1288.

20. Tortopidis D, Lyons MF, Baxendale RH, Gilmour WH. The variability of bite force measurement between sessions, in different positions within the dental arch. *J Oral Rehabil* 1998;**25:**681–686.
21. Shinogaya T, Bakke M, Thomsen CE, Vilmann A, Matsumoto M. Bite force and occlusal load in healthy young subjects–a methodological study. *Eur J Prosthodont Restor Dent* 2000;**8:**11–15.
22. Ferrario VF, Sforza C, Serrao G, Dellavia C, Tartaglia GM. Single tooth bite forces in healthy young adults. *J Oral Rehabil* 2004;**31:**18–22.
23. Sodek J, McKee MD. Molecular and cellular biology of alveolar bone. *Periodontol 2000* 2000;**24:**99–126.
24. Wesselink PR, Beertsen W. Repair processes in the periodontium following dentoalveolar ankylosis: the effect of masticatory function. *J Clin Periodontol* 1994;**21:**472–478.
25. Andreasen JO. External root resorption: its implication in dental traumatology, paedodontics, periodontics, orthodontics and endodontics. *Int Endod J* 1985;**18:** 109–118.
26. Ten Cate AR. *Histología oral. Desarrollo, estructura y función*. Buenos Aires: Ed. Panamericana, 1986.
27. Gómez de Ferraris ME, Campos Muñoz A. *Histología y Embriología Bucodental*. Madrid: Ed. Panamericana, 2002.
28. Andreasen JO. Review of root resorption systems and models. Etiology of root resorption and the homeostatic mechanisms of periodontal ligament. In: Davidovitch Z, ed. *The biological Mechanisms of Tooth Eruption and Root Resorption*. Birmingham (AL): EBSCO Media, 1988:9–21.
29. Ishigaki S, Kurozumi T, Morishige E, Yatani H. Occlusal interference during mastication can cause pathological tooth mobility. *J Periodontal Res* 2006;**41:**189–192.
30. Brudvik P, Rygh P. The repair of orthodontic root resorption: an ultrastructural study. *Eur J Orthod* 1995;**17:**189–198.
31. Kurol J, Owman-Moll P. Hyalinization and root resorption during early orthodontic tooth movement in adolescents. *Angle Orthod* 1998;**68:**161–165.
32. Davidovitch Z. Tooth movement. *Crit Rev Oral Biol Med* 1991;**2:**411–450.
33. Grieve WG 3rd, Johnson GK, Moore RN, Reinhardt RA, DuBois LM. Prostaglandin E (PGE) and interleukin-1 beta (IL-1 beta) levels in gingival crevicular fluid during human orthodontic tooth movement. *Am J Orthod Dentofacial Orthop* 1994;**105:**369–374.
34. Boekenoogen DI, Sinha PK, Nanda RS, Ghosh J, Currier GF, Howes RI. The effects of exogenous prostaglandin E2 on root resorption in rats. *Am J Orthod Dentafacial Orthop* 1996;**109:**277–286.
35. Uematsu S, Mogi M, Deguchi T. Interleukin (IL)-1 beta, IL-6, tumor necrosis factor-alpha, epidermal growth factor, and beta 2-microglobulin levels are elevated in gingival crevicular fluid during human orthodontic tooth movement. *J Dent Res* 1996;**75:**562–567.
36. Uematsu S, Mogi M, Deguchi T. Increase of transforming growth factor-beta 1 in gingival crevicular fluid during human orthodontic tooth movement. *Arch Oral Biol* 1996;**41:**1091–1095.
37. Nakaya H, Oates TW, Hoang AM, Kamoi K, Cochran DL. Effects of interleukin-1 beta on matrix metalloproteinase-3 levels in human periodontal ligament cells. *J Periodontol* 1997;**68:**517–523.
38. Giargia M, Lindhe J. Tooth mobility and periodontal disease. *J Clin Periodontol* 1997;**24:**785–795.
39. Shimizu N, Ozawa Y, Yamaguchi M, Goseki T, Ohzeki K, Abiko Y. Induction of COX-2 expression by mechanical tension force in human periodontal ligamentcells. *J Periodontol* 1998;**69:**670–677.
40. Chien HH, Lin WL, Cho ML. Interleukin-1B induced release of matrix proteins into culture media causes inhibition of mineralization of nodules formed by periodontal ligament cells *in vitro*. *Calcif Tissue Int* 1998;**64:**402–413.
41. Tzannetou S, Efstratiadis S, Nicolay O, Grbic J, Lamster I. Interleukin-1beta and beta-glucuronidase in gingival crevicular fluid from molars during rapid palatal expansion. *Am J Orthod Dentofacial Orthop* 1999;**115:**686–696.
42. Iwasaki LR, Haack JE, Nickel JC, Reinhardt RA, Petro TM. Human interleukin-1 beta and interleukin-1 receptor antagonist secretion and velocity of tooth movement. *Arch Oral Biol* 2001;**46:** 185–189.
43. Alhashimi N, Frithiof L, Brudvik P, Bakhiet M. Orthodontic tooth movement and de novo synthesis of proinflammatory cytokines. *Am J Orthod Dentofacial Orthop* 2001;**119:**307–312.
44. Glickman I, Smulow JB. Alterations in the pathway of gingival inflammation into the underlyng tissues induced by excessive occlusal forces. *J Periodontol* 1962;**33:** 7–13.
45. Glickman I, Smulow JB. Effect of excessive occlusal forces on the pathway of gingival inflammation in humans. *J Periodontol* 1965;**36:**141–147.
46. Glickman I, Smulow JB. The combined effects of inflammation and trauma from occlusion in periodontitis. *Int Dent J* 1969;**19:**393–407.
47. Stahl SS. Accommodation of the periodontium to occlusal trauma and inflammatory periodontal disease. *Dent Clin North Am* 1975;**19:**531–542.
48. Gher ME. Changing concepts. The effects of occlusion on periodontitis. *Dent Clin North Am* 1998;**42:**285–299.
49. Harrel SK. Occlusal forces as a risk factor for periodontal disease. *Periodontol 2000* 2003;**32:**111–117.
50. Rodríguez Pato R. Reabsorciones radiculares en la periodontitis del adulto. Tesis Doctoral. Universidad de Santiago de Compostela. Facultad de Medicina. 2002.
51. Johansson AS, Svensson KG, Trulsson M. Impaired masticatory behavior in subjects with reduced periodontal tissue support. *J Periodontol* 2006;**77:**1491–1497.
52. Takeuchi N, Yamamoto T. Correlation between periodontal status and biting force in patients with chronic periodontitis during the maintenance phase of therapy. *J Clin Periodontol* 2008;**35:**215–220.